PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION.

DISSERTATION SUBMITTED FOR
MD DEGREE (BRANCH 1) GENERAL MEDICINE
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THE TAMILNADU DR.M.G.R.MEDICAL
UNIVERSITY
CHENNAI,TAMILNADU.

CERTIFICATE

This is to certify that this dissertation titled "PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION" submitted by DR.C.R.MAHESH BABU to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree branch I General Medicine, is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, Dr.C.R.MAHESH BABU , solemnly declare that the dissertation titled "PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION"- AN OBSERVATION STUDY has been prepared by me. This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the regulations for the award of MD degree (branch I) General Medicine.

Place: Madurai

Date: Dr. C.R.MAHESH BABU.

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CONTENTS

INTRODUCTION

INTRODUCTION

The metabolic syndrome is one of the major public health issues of this century. It is a constellation of physical conditions and metabolic abnormalities, commonly occurring together, that increases an individual's risk for development of type 2 diabetes mellitus and cardiovascular disease. If the current trend continues, the premature deaths and disabilities resulting from these conditions will increase the financial burden in developing countries.

Several expert groups have attempted to define the diagnostic criteria for the metabolic syndrome. In 1998, the World Health Organization (WHO) proposed a formal definition of the metabolic syndrome; according to this, a person must have either glucose intolerance or insulin resistance along with two of the following four criteria: central obesity, hypertension, dyslipidemia, and albuminuria. In 2001, the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III)[4] provided a new definition for the metabolic syndrome, according to which a person must have three of the following five abnormalities: abdominal adiposity, hypertension, hypertriglyceridemia, low high-density lipoprotein cholesterol, and elevated fasting glucose. Based on the definitions of NCEP ATP III, a cross-sectional study conducted by the Third National Health and Nutrition Examination Survey in a US population found that the prevalence of the syndrome was 25% among white Americans; the prevalence was 44% among those 50 years and older.

More recent estimates of the prevalence of the metabolic syndrome ranged from 21.3-32.8% among the participants in the Framingham Offspring Study and the San Antonio Heart Study [6]. Moreover, a recent study in patients with established coronary artery disease or stroke showed that the prevalence of the metabolic syndrome correlated with the extent of vascular damage. Little is known about the prevalence of the metabolic syndrome in patients with acute coronary syndrome, particularly in South-East Asians. There is also limited information available on the impact of the metabolic syndrome on hospital outcomes after presentation for an acute myocardial infarction (AMI) in South-East Asians.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

THE METABOLIC SYNDROME:

In 1988, Gerald Reaven[11] reintroduced the concept of syndrome X for the clustering of cardiovascular risk factors like hypertension, glucose intolerance, high triglycerides, and low HDL cholesterol concentrations. The syndrome is however, much older, having been already observed in 1923 by Kylin, who described the clustering of hypertension, hyperglycemia, and gout as a syndrome. Subsequently, several other metabolic abnormalities have been associated with this syndrome, including obesity, microalbuminuria, and abnormalities in fibrinolysis and coagulation. The syndrome has also been given several other names, including the metabolic syndrome, the insulin resistance syndrome, the plurimetabolic syndrome, and the deadly quartet. The name “insulin resistance syndrome” has been widely used and refers to insulin resistance as a common denominator of the syndrome. The prevalence of the metabolic syndrome has varied markedly between different studies, most likely because of the lack of fixed criteria for the definition of the syndrome. In 1998, WHO proposed a unifying definition for the syndrome and chose to call it the metabolic syndrome rather than the insulin resistance syndrome. This name was chosen primarily because it was not considered established that insulin resistance was the cause of all the components of the syndrome.

The metabolic syndrome (syndrome X, insulin resistance syndrome) consists of a constellation of metabolic abnormalities that confer increased risk of cardiovascular disease (CVD) and diabetes mellitus (DM). The criteria for the metabolic syndrome have evolved since the original definition by the World Health Organization in 1998, reflecting growing clinical evidence and analysis by a variety of consensus conferences and professional organizations. The major features of the metabolic syndrome include central obesity, hypertriglyceridemia, low HDL cholesterol, hyperglycemia, and hypertension.

Epidemiology

Prevalence of the metabolic syndrome varies across the globe, in part reflecting the age and ethnicity of the populations studied and the diagnostic criteria applied. In general, the prevalence of metabolic syndrome increases with age. The highest recorded prevalence worldwide is in Native Americans,

with nearly 60% of women ages 45–49 and 45% of men ages 45–49 meeting National Cholesterol Education Program, Adult Treatment Panel III (NCEP:ATPIII) criteria. Based on data from the National Health and Nutrition Examination Survey (NHANES) III[5], the age-adjusted prevalence of the metabolic syndrome in the United States is 34% for men and 35% for women. In France, a 30–64-year-old cohort shows a <10% prevalence for each gender, although 17.5% are affected in the 60–64 age range. Greater industrialization worldwide is associated with rising

rates of obesity, which is anticipated to dramatically increase the prevalence of the metabolic syndrome, especially as the population ages. Moreover, the rising prevalence and severity of obesity in children is initiating features of the metabolic syndrome in a younger population.

Risk Factors

Overweight / Obesity

Central adiposity is a key feature of the syndrome, reflecting the fact that the syndrome's prevalence is driven by the strong relationship between waist circumference and increasing adiposity. However, despite the importance of obesity, patients who are normal weight may also be insulin-resistant and have the syndrome.

Sedentary Lifestyle

Physical inactivity is a predictor of CVD events and related mortality. Many components of the metabolic syndrome are associated with a sedentary lifestyle, including increased adipose tissue (predominantly central); reduced HDL cholesterol; and a trend toward increased triglycerides, high blood pressure, and high blood glucose levels in the genetically susceptible. Compared with individuals who watched television or videos or used their computer <1 hour daily, those who

carried out these behaviors for >4hours daily have a twofold increased risk of the syndrome.

<table>
<tr><th colspan="4">NCEP:ATPIII 2001 and IDF Criteria for the Metabolic Syndrome</th></tr>
<tr><td>NCEP:ATPIII 2001</td><td colspan="3">IDF Criteria for Central Adiposity</td></tr>
<tr><td>Three or more of the following:</td><td colspan="3">Waist Circumference</td></tr>
<tr><td rowspan="8">Central obesity: Waist circumference >102 cm (M), >88 cm (F)

Hypertriglyceridemia: Triglycerides 150 mg/dL or specific medication

Low HDL cholesterol: <40 mg/dL and <50 mg/dL, respectively, or specific medication

Hypertension: Blood pressure 130 mm systolic or 85 mm diastolic or specific medication

Fasting plasma glucose 100 mg/dL or specific medication or previously diagnosed type 2 diabetes</td><td>Men</td><td>Women</td><td>Ethnicity</td></tr>
<tr><td>94 cm</td><td>80 cm</td><td>Europid, Sub-Saharan African, Eastern & Middle Eastern</td></tr>
<tr><td>90 cm</td><td>80 cm</td><td>South Asian, Chinese, and ethnic South & Central American</td></tr>
<tr><td>85 cm</td><td>90 cm</td><td>Japanese</td></tr>
<tr><td colspan="3">Two or more of the following:</td></tr>
<tr><td colspan="3">Fasting triglycerides >150 mg/dL or specific medication</td></tr>
<tr><td colspan="3">HDL cholesterol <40 mg/dL and <50 mg/dL for men and women, respectively, or specific medication</td></tr>
<tr><td colspan="3">Blood pressure >130 systolic or >85 mm diastolic or previous diagnosis or specific medication</td></tr>
<tr><td colspan="3">Fasting plasma glucose 100 mg/dL or previously diagnosed type 2 diabetes</td></tr>
</table>

Ageing

The metabolic syndrome affects 44% of the U.S. population older than age 50. A greater percentage of women older than age 50 have the syndrome than men. The age dependency of the syndrome's prevalence is seen in most populations around the world.

Diabetes Mellitus

DM is included in both the NCEP[4] and International Diabetes Foundation (IDF) definitions of the metabolic syndrome. It is estimated that the large majority (~75%) of patients with type 2 diabetes or impaired glucose tolerance (IGT) have the metabolic syndrome. The presence of the metabolic syndrome in these populations relates to a higher prevalence of CVD compared to patients with type 2 diabetes or IGT without the syndrome.

Coronary Heart Disease

The approximate prevalence of metabolic syndrome in patients with coronary heart disease (CHD)[7,9] is 50%, with a prevalence of 37% in patients with premature coronary artery disease (age 45), particularly in women. With appropriate cardiac rehabilitation and changes in lifestyle (e.g., nutrition, physical activity, weight reduction, and, in some cases, pharmacologic agents), the prevalence of the syndrome can be reduced.

Lipodystrophy

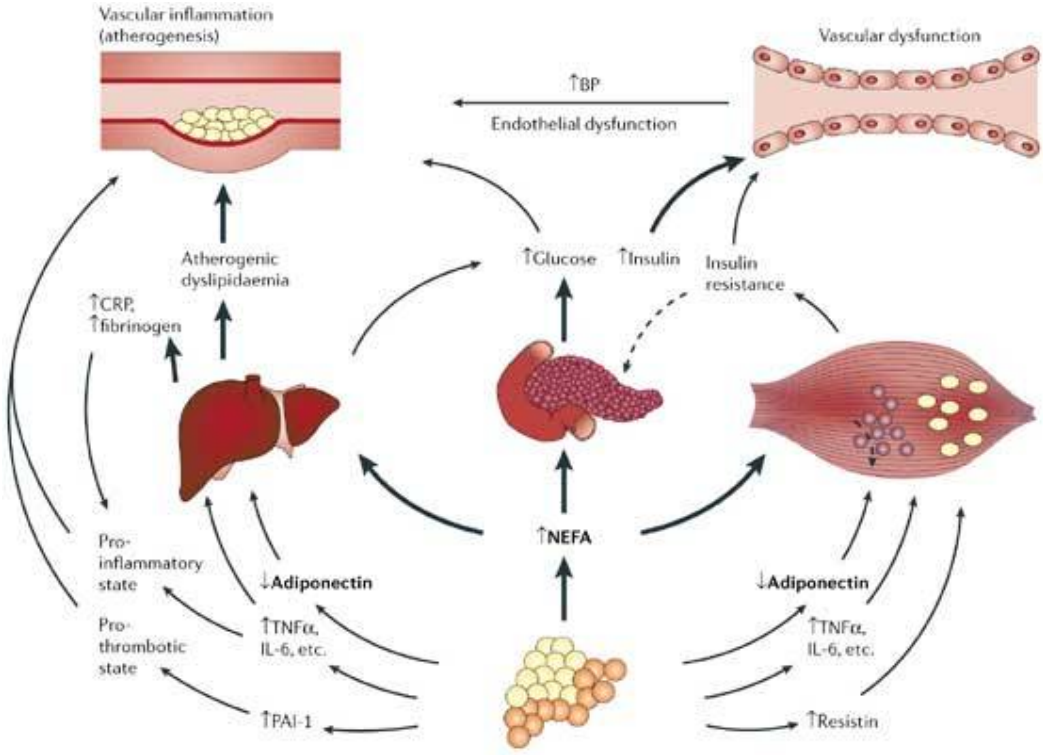
Lipodystrophic disorders in general are associated with the metabolic syndrome. Both genetic (e.g., Berardinelli-Seip congenital lipodystrophy, Dunnigan familial partial lipodystrophy) and acquired (e.g., HIV-related lipodystrophy in patients treated with highly active antiretroviral therapy) forms of lipodystrophy may give rise to severe insulin resistance and many of the metabolic syndrome's components.

Etiology

Insulin Resistance

The most accepted and unifying hypothesis to describe the pathophysiology of the metabolic syndrome is insulin resistance, caused by an incompletely understood defect in insulin action . The onset of insulin resistance is heralded by

postprandial hyperinsulinemia, followed by fasting hyperinsulinemia and, ultimately, hyperglycemia.



An early major contributor to the development of insulin resistance is an overabundance of circulating fatty acids. Plasma albumin-bound free fatty acids (FFAs) are derived predominantly from adipose tissue triglyceride stores released by hormone-sensitive lipase. Fatty acids are also derived through the lipolysis of triglyceride-rich lipoproteins in tissues by lipoprotein lipase (LPL). Insulin mediates both antilipolysis and the stimulation of LPL in adipose tissue. Of note, the inhibition of lipolysis in adipose tissue is the most sensitive pathway of insulin action. Thus, when insulin resistance develops, increased lipolysis produces more

fatty acids, which further decrease the antilipolytic effect of insulin. Excessive fatty acids enhance substrate availability and create insulin resistance by modifying downstream signaling. Fatty acids impair insulin-mediated glucose uptake and accumulate as triglycerides in both skeletal and cardiac muscle, whereas increased glucose production and triglyceride accumulation are seen in liver.

The oxidative stress hypothesis provides unifying theory for aging and the predisposition to the metabolic syndrome. In studies carried out in insulin-resistant subjects with obesity or type 2 diabetes, in the offspring of patients with type 2 diabetes, and in the elderly, a defect has been identified in mitochondrial oxidative phosphorylation, leading to the accumulation of triglycerides and related lipid molecules in the muscle. The accumulation of lipids in muscle is associated with insulin resistance.

Increased Waist Circumference

Waist circumference is an important component of the most recent and frequently applied diagnostic criteria for the metabolic syndrome. However, measuring waist circumference does not reliably distinguish between a large waist due to increases in subcutaneous adipose tissue against visceral fat; this distinction requires CT or MRI. With increases in visceral adipose tissue, adipose tissue-derived FFAs are directed to the liver. On the other hand, increases in abdominal subcutaneous fat release lipolysis products into the systemic circulation

and avoid more direct effects on hepatic metabolism. Relative increases in visceral versus subcutaneous adipose tissue with increasing waist circumference in Asians and Asian Indians may explain the greater prevalence of the syndrome in these populations compared to African-American men in whom subcutaneous fat predominates. It is also possible that visceral fat is a marker for, but not the source of, excess postprandial FFAs in obesity.

Standardized instructions for the measurement of waist circumference

- It is preferable to use a tape with a spring handle in order to control the tension applied on the abdomen (e.g. Gulick model).
- Otherwise, use an unstretchable tape (avoid fabric tape) with an ungraduated extremity of 3-5 cm in order to properly grab the tape.

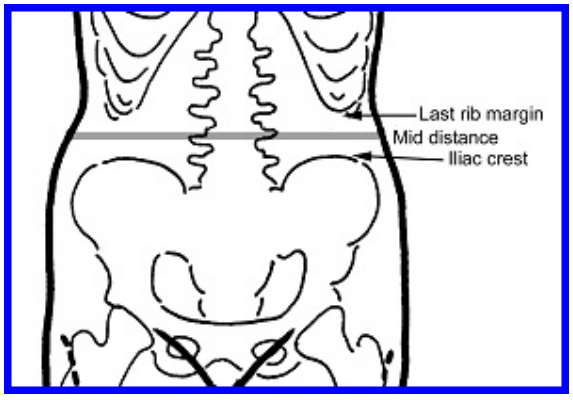
The subject stands with his feet shoulder-width apart. The arms hang on each side of the body but out at an angle of about 30 degrees to allow the person taking the measurement to work comfortably. If this is not comfortable, alternatively participants can cross their arms on their shoulders in a relaxed manner. A slight tension should be applied to the tape (until the red mark appears) at the moment of the reading.

The measurement is taken at the end of a normal expiration, while ensuring that the participant does not contract the abdominal muscles. (Experimenter can

engage conversation with patient if he is suspected to contract the abdominal muscles). The measurement is made twice and a third time if the difference between the first two measurements is greater than 5% (+/- 1 cm). The two closest measurements will be averaged.

Detailed instructions

1. Mark with a pencil bony landmarks of the right and left last rib margin.
2. Mark with a pencil bony landmarks of the right and left iliac crest.
3. Mark with a pencil the mid-distance between the last rib margin and the top of the iliac crest of the two sides.
4. Measure mid way between the two bony points 1 and 2.



Dyslipidemia

In general, FFA flux to the liver is associated with increased production of apoB–containing, triglyceride-rich very low density lipoproteins (VLDLs). The effect of insulin on this process is complex, but hypertriglyceridemia is an excellent marker of the insulin-resistant condition.

The other major lipoprotein disturbance in the metabolic syndrome is a reduction in HDL cholesterol. This reduction is a consequence of changes in HDL composition and metabolism. In the presence of hypertriglyceridemia, a decrease in the cholesterol content of HDL is a consequence of reduced cholesteryl ester content of the lipoprotein core in combination with cholesteryl ester transfer protein–mediated alterations in triglyceride making the particle small and dense. This change in lipoprotein composition also results in an increased clearance of HDL from the circulation. The relationships of these changes in HDL to insulin resistance are likely indirect, occurring in concert with the changes in triglyceride-rich lipoprotein metabolism.

In addition to HDL, LDLs are also modified in composition. With fasting serum triglycerides >180 mg/dL, there is almost always a predominance of small dense LDLs. Small dense LDLs are thought to be more atherogenic. They may be toxic to the endothelium, and they are able to transit through the endothelial basement membrane and adhere to glycosaminoglycans. They also have

increased susceptibility to oxidation and are selectively bound to scavenger receptors on monocyte-derived macrophages. Subjects with increased small dense LDL particles and hypertriglyceridemia also have increased cholesterol content of both VLDL1 and VLDL2 subfractions. This relatively cholesterol rich VLDL particle may also contribute to the atherogenic risk in patients with metabolic syndrome.

Glucose Intolerance

The defects in insulin action lead to impaired suppression of glucose production by the liver and kidney and reduced glucose uptake and metabolism in insulin-sensitive tissues, i.e., muscle and adipose tissue. The relationship between impaired fasting glucose (IFG) or impaired glucose tolerance (IGT) and insulin resistance is well supported by human, nonhuman primate, and rodent studies. To compensate for defects in insulin action, insulin secretion and/or clearance must be modified to sustain euglycemia. Ultimately, this compensatory mechanism fails, usually because of defects in insulin secretion, resulting in progress from IFG and/or IGT to DM.

Hypertension

The relationship between insulin resistance and hypertension is well established. Paradoxically, under normal physiologic conditions, insulin is a vasodilator with secondary effects on sodium reabsorption in the kidney. However,

in the setting of insulin resistance, the vasodilatory effect of insulin is lost, but the renal effect on sodium reabsorption is preserved. Insulin also increases the activity of the sympathetic nervous system, an effect that may also be preserved in the setting of the insulin resistance. Finally, insulin resistance is characterized by pathway-specific impairment in phosphatidylinositol 3-kinase signaling. In the endothelium, this may cause an imbalance between the production of nitric oxide and secretion of endothelin-1, leading to decreased blood flow. Although these mechanisms are provocative, when insulin action is assessed by levels of fasting insulin or by the Homeostasis Model Assessment (HOMA), insulin resistance contributes only modestly to the increased prevalence of hypertension in the metabolic syndrome.

Proinflammatory Cytokines

The increases in proinflammatory cytokines, including interleukin (IL) 1, IL-6, IL-18, resistin, tumor necrosis factor (TNF), and C-reactive protein (CRP), reflect overproduction by the expanded adipose tissue mass. Adipose tissue-derived macrophages may be the primary source of pro-inflammatory cytokines locally and in the systemic circulation. It remains unclear, however, how much of the insulin resistance is caused by the paracrine versus endocrine effects of these cytokines.

Adiponectin

Adiponectin is an anti-inflammatory cytokine produced exclusively by adipocytes. Adiponectin enhances insulin sensitivity and inhibits many steps in the inflammatory process. In the liver, adiponectin inhibits the expression of gluconeogenic enzymes and the rate of glucose production. In muscle, adiponectin increases glucose transport and enhances fatty acid oxidation, partially due to activation of AMP kinase. Adiponectin is reduced in the metabolic syndrome. The relative contribution of adiponectin deficiency and overabundance of the proinflammatory cytokines remains unclear.

MYOCARDIAL INFARCTION

Myocardial infarction is often depicted as a modern disease it was clearly recognized before the modern era by Morgagni in 1761. Dr.Williiam Heberden was the first person to describe Angina Pectoris when he described it at a meeting of the Royal college of Physicians in 1768. Dr.John Wall of Worchester was the first to ascribe angina to heart disease. An early personal description of myocardial infarction was given by the famous surgeon sir John Hunter who himself experienced what was probably a MI in 1773. The description of his subsequent autopsy describes the scarred areas in his heart. Dr.John Fothergill in 1773 described a small white cicatrix "as big as six pence" near the apex of the ventricle . In the later years four young men Jenner, Perry, Black and Burn

established the "Ischaemic theory of Angina" with an autopsy showing a clot in the coronary. It was approximately 120 years late the leaders of Medical Profession generally accepted the concept. The first ever description of a nonfatal MI comes from V.P.Obraztor and N.D.Stratzhesko of erst while U.S.S.R. in 1910. Americans Physicians attribute the same achievement to James B.Herrick a Chicago physician who in 1912 in the journal of the American Medical Association described the clinical features of sudden obstruction of the coronary arteries. Both these reports belied the then current opinion that MI was universally fatal. Since then the Medical community has travelled far and wide into this subject.

Coronary Atherosclerosis

Atherosclerosis develops focally in time as well as space. Atherosclerosis occurs over many years, usually many decades. The growth of atherosclerotic plaque probably does not occur in a smooth linear fashion but rather discontinuously. The clinical expression may be chronic as in stable effort angina or a much more dramatic incident as in myocardial infarction. Fatty streaks represent the initial lesion of atherosclerosis. Lipoproteins accumulate in the intima of the arteries by binding to proteoglycans of extracellular matrix. Sequestration within the intima seperates the lipoprotein from plasma anti-oxidants leading to oxidative modification. This may trigger a local inflammatory response signaling the subsequent steps. Next step is adhesion of mononuclear leucocytes

to luminal endothelium followed by migration to intima. A number of leucocyte adhesion molecules, various cytokines, even modified lipoproteins mediate this response. Once within the intima, mononuclear phagocytes differentiate to macrophages and imbibes lipid and transform to lipid laden foam cells. The uptake of lipids by macrophages is not via the classical LDH receptor but by the scavenger receptor. Some of the foam cells undergo apoptosis leading to the central necrotic core. The fatty streak evolves to a complicated atherosclerotic lesion with accumulation of smooth cells and extracellular matrix elaborated by smooth muscle cells of intima overlies the lipid rich core. Plaques that are vulnerable to rupture have thin fibrous cap relatively large lipid cores and a high content of macrophages. The sites of plaque rupture have fewer number of smooth muscle cells.

Pathophysiology – Role of Acute Plaque Rupture

MI generally occurs when coronary blood flows decreases abruptly after occlusion of a coronary artery previously affected by atherosclerosis. In most cases mi occurs when an atherosclerotic plaque fissures, ruptures or ulcerates and when condition favour thrombogenesis, so that a mural thrombus is formed leading to coronary artery occlusion. After an initial platelet monolayer forms at the site of ruptured plaque various agonists (collagen, ADP, epinephrine,serotonin) promote platelet activation . After activation, platelets release thromboxane A2 causing

further platelet activation and potential resistance to thrombolysis. Platelet activation also leads to conformational change to glycoprotein IIb / IIIa receptor causing high affinity in vWF & fibrinogen. The coagulation cascade is activated on exposure to tissue factor at the site of plaque rupture. Factor VII and X are activated ultimately leading to conversion of prothrombin to thrombin. The culprit artery eventually becomes occluded by a thrombus containing platelet aggregates and fibrin strands. Rarely MI may be due to coronary artery occlusion by emboli, congenital anomalies, coronary spasm or due to systemic inflammatory disease.

Risk factors for CAD

Risk factors for CAD can be broadly divided into modifiable and nonmodifiable. The modifiable one can be divided into lipid factors and nonlipid factors. The nonlipid risk factors include DM, HTN, smoking, positive family history, gender, BMI, waist hip ratio, mental stress, while the lipid risk factors include total cholesterol level, TGL level, low level of HDL- C and high level of LDL-C. Novel risk factors include CRP, homocysteine, high plasma fibrinogen, high plasminogen activator inhibitor, lipoprotein(a), lipoprotein subclass like small oxidized LDL. It was Virchow who recognized the role of lipids is atherosclerosis. Before 1950, there was no clear perception of the interrelationship of serum lipids, atherosclerosis and CAD. Since then research laboratories have made conflicting claims for the most useful measurement of serum lipids levels in detecting and

managing CAD. Emphasis has been placed in turn on the measurement of levels of serum cholesterol, lipoproteins and TGL. Physical separation and characterization of serum lipoproteins by ultra centrifugation and electrophoresis resulted in two classifications system for lipoproteins based on density and electrophoretic mobility respectively.

RISK FACTORS FOR CAD

Non-modifiable

1. Age
2. Family history of premature atherosclerosis*
3. Male sex

Modifiable, under study

1. Alcohol intake (other than moderate)
2. *Chlamydia pneumoniae* infection
3. High CRP level
4. High level of small, dense LDL
5. High lipoprotein(a) level
6. Hyperhomocysteinemia
7. Hyperinsulinemia
8. Hypertriglyceridemia
9. 5-Lipoxygenase polymorphisms
10. Low intake of fruits and vegetables
11. Obesity or metabolic syndrome
12. Prothrombotic states (eg, hyperfibrinogenemia, high plasminogen activator inhibitor level)
13. Psychosocial factors (eg, type A personality, depression, anxiety, work characteristics, socioeconomic status)
14. Renal insufficiency
15. Sedentary lifestyle

Modifiable, established

1. Certain dyslipidemias (high total or LDL level, low HDL level, increased total-to-HDL cholesterol ratio)
2. Cigarette smoking
3. Diabetes mellitus
4. Hypertension

AIM OF THE STUDY

AIM OF THE STUDY.

1. To ascertain the prevalence of the metabolic syndrome in patients with acute myocardial infarction.
2. To study the impact of the metabolic syndrome on hospital outcomes.
3. To find out the association of each component of the metabolic syndrome with acute myocardial infarction.

MATERIALS AND METHODS

MATERIALS AND METHODS

SETTING: Acute Myocardial infarction patients admitted to medical wards and cardiac ICU at Govt Rajaji Hospital, Madurai.

COLLABORATING DEPARTMENTS:

1. Department of cardiology,
   Madurai medical college,
   Madurai.
2. Department of bio – chemistry,
   Madurai medical college,
   Madurai.

DESIGN OF STUDY: Prospective analytic study

PERIOD OF STUDY: 01.04.2010 TO 30.09.2010

SAMPLE SIZE: 100

SELECTION OF STUDY SUBJECTS: 100 consecutive patients admitted with acute myocardial infarction in the department of medicine and cardiology ICU from 01.04.2010 to 30.09.2010 formed the study group. Patients who did not satisfy the criteria for metabolic syndrome served as the control group.

Blood samples were drawn the following morning for HDL, TGL, and FBS. Waist circumference was measured and BP was recorded in all patients.

Patients were diagnosed as having the metabolic syndrome if they had any three of the following five components:

1. Abdominal obesity (waist circumference >102 cm in men and >88 cm in women)
2. High triglyceride levels (≥150 mg/dl)
3. Low HDL-C levels (<40 mg/dl in men and <50 mg/dl in women)
4. Elevated fasting glucose levels (≥110 mg/dl)
5. High blood pressure (treated hypertension and systolic blood pressure/diastolic blood pressure ≥130/85 mm Hg).

Data was analyzed using the Student's t test and the Chi-square test.

INCLUSION CRITERIA:

All patients with myocardial infarction admitted in medical wards and cardiology ICU were taken.

EXCLUSION CRITERIA:

Patients with secondary causes affecting lipid profile were excluded.,

1. Hypothyroidism
2. Nephrotic syndrome

3. Chronic kidney disease
4. Cushing syndrome
5. Oral contraceptives
6. Patients not willing to participate in the study.

ETHICAL APPROVAL: Obtained

CONSENT: informed consent was obtained

FINANCIAL SUPPORT: NIL

CONFLICT OF INTEREST: NIL

RESULTS

STATISTICAL ANALYSIS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2002).

Using this software, range, frequencies, percentages, means and standard deviations were calculated.

Sensitivity, specificity, accuracy, positive predictive value and negative predictive values were calculated using the following formulae

$$\text{Sensitivity} = \frac{\text{True positive}}{\text{True positive + False negative}} \times 100$$

$$\text{Specificity} = \frac{\text{True negative}}{\text{False positive + True negative}} \times 100$$

$$\text{Positive predictive value} = \frac{\text{True positive}}{\text{True positive + False positive}} \times 100$$

$$\text{Negative predictive value} = \frac{\text{True negative}}{\text{True negative + False negative}} \times 100$$

$$\text{Accuracy} = \frac{\text{True positive + True negative}}{N}$$

A.PROFILE OF CASES STUDIED:

100 patients who were admitted with acute myocardial infarction were selected and following parameters were analysed which are as follows.

TABLE 1

QUANTITATIVE PARAMETERS

PARAMETER	RANGE	MEAN	SD
AGE (in years)	33 - 70	56.1	6.4
EF (%)	30 – 45	38.9	3.4
DURATION OF STAY(days)	2 – 15	8.3	2.6
SBP (mmHg)	80 – 190	125.1	22.5
DBP (mmHg)	60 – 110	80.7	11.2
HDL (mg%)	36 – 52	43.8	4.0
TGL (mg%)	100 – 460	166.7	70.0
FBS (mg%)	72 – 336	123	45.0
AC (in cm)	73 – 100	92.2	10.7

TABLE 2

QUALITATIVE PARAMETERS.

PARAMETER		CASES	
		NUMBER	PERCENTAGE
GENDER	MALES	87	87
	FEMALES	13	13
AREA	RURAL	69	69
	URBAN	31	31
OCCUPATION	ACTIVE	35	35
	SEDENTARY	65	65
EXERCISE	YES	45	45
	NO	55	55
SMOKING (MALES)	YES	72	82.8
	NO	15	17.2
DM	YES	65	65
	NO	35	35
HT	YES	76	76
	NO	24	24
FAMILY HISTORY	YES	77	77
	NO	23	23
DIET	VEGETARIAN	33	33
	NON VEGETARIAN	67	67
ALCOHOLS (MALES)	YES	38	43.7
	NO	49	56.3
S / N	STEMI	94	94
	NSTEMI	6	6
MI	AWMI	79	79
	IWMI	21	21
KILLIP CLASS	1	21	21
	2	58	58
	3	18	18
	4	3	3
THROMBOLYSIS	THROMBOLYSED	91	91
	NOT THROMBOLYSED	9	9
D / A	DEAD	4	4
	ALIVE	96	96

Among the 100 patients 87 were males and 13 were females. 69 patients were from rural area and 31 patients were from urban area. 35 were doing active work and 65 were doing sedentary job. 45 were doing regular exercises and 55 had no habit of exercising. Among the 87 male patients 72 were smokers. None of the females were smokers. 38 male patients were used to consume alcohol and none of the females took alcohol. Diabetes was pre-existing in 65 patients and hypertension was pre-existing in 76 patients. Family history of either DM/ HTN/ CAD was present in 77 patients. 67 patients were taking mixed diet and remaining 33 were taking vegetarian diet. STEMI occurred in 94 patients and NSTEMI occurred in 6 patients. AWMI occurred in 79 patients and IWMI occurred in 21 patients. 58 patients presented in KILLIP class 2. Only 3 patients presented in class 3. Thrombolysis was done in 91 patients and in the remaining 9 patients it was not done due to contraindications. Among the 100 patients 4 of them died during the hospital stay.

TABLE 3

PREVALENCE OF METABOLIC SYNDROME

METABOLIC SYNDROME	CASES	
	NUMBER	PERCENTAGE
POSITIVE	32	32
NEGATIVE	68	68
TOTAL	100	100

Among the 100 patients 32 patients satisfied the criteria for metabolic syndrome and taken as study subjects and the remaining 78 patients formed the internal control group. Further comparisons were made between these two groups.

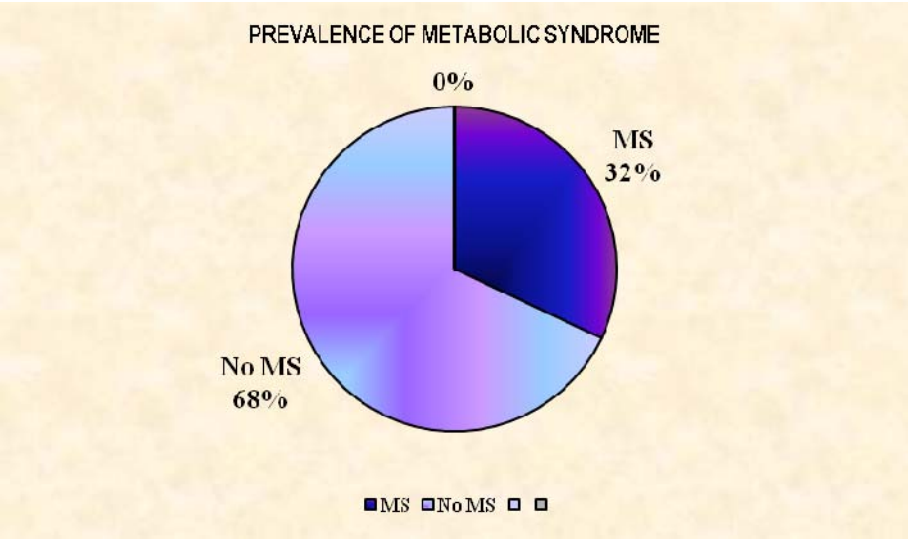


TABLE 4

OUTCOME IN METABOLIC SYNDROME

METABOLIC SYNDROME	OUTCOME			
	ALIVE		DEAD	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
POSITIVE (32)	29	90.6	3	9.4
NEGATIVE(68)	67	98.5	1	1.5
' p'	0.0952 NOT SINIFICANT			

Among the 32 patient in the study group 3(9.4%) patients died and among the 78 patients in the control group 1(1.5%) patient died and the 'p' value was 0.0952 which was statistically not significant.

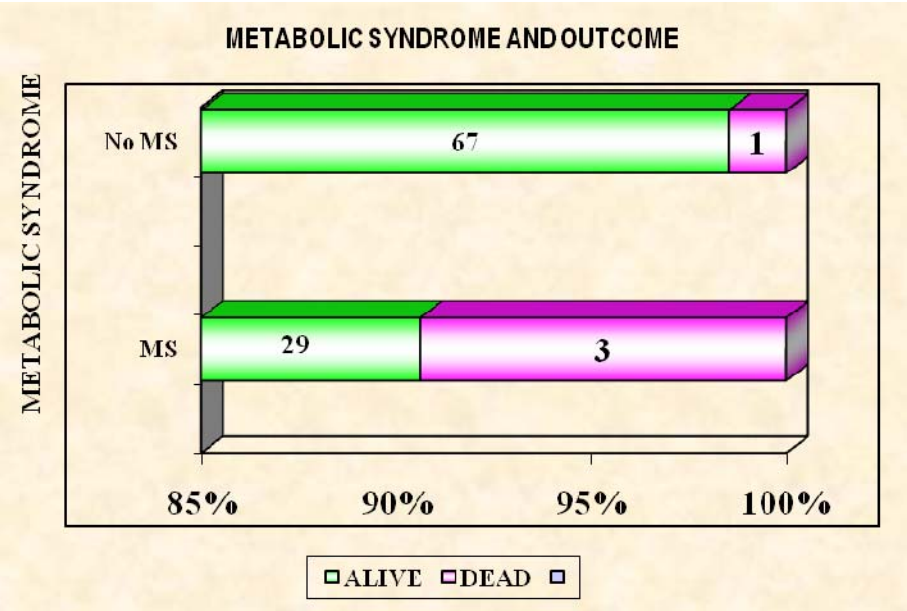


TABLE 5

ASSOCIATION BETWEEN FIVE COMPONENTS OF METABOLIC SYNDROME

COMPONENTS OF METABOLIC SYNDROME		TOTAL CASE	METABOLIC SYNDROME				'p'
			POSITIVE		NEGATIVE		
B . P	POSITIVE	80	29	36.3	51	63.8	0.1201 (NOT SIGNIFICANT)
	NEGATIVE	20	3	15	17	85	
HDL	ABNORMAL	21	15	71.4	6	28.6	0.0001 (SIGNIFICANT)
	NORMAL	79	17	21.5	62	78.5	
TGL	ABNORMAL	49	30	61.2	19	38.8	0.0001(SIGNIFICANT)
	NORMAL	51	2	3.9	49	96.1	
FBS	ABNORMAL	36	29	80.6	7	19.4	0.0001(SIGNIFICANT
	NORMAL	64	3	4.7	61	95.3	
AC	ABNORMAL	27	24	88.9	3	11.1	0.0001(SIGNIFICANT)
	NORMAL	73	8	11	65	89	

The association between BP and metabolic syndrome was not significant statistically as the 'p' value was 0.1201. There was significant statistical association between HDL, TGL, FBS, AC and metabolic syndrome (p value – 0.0001).

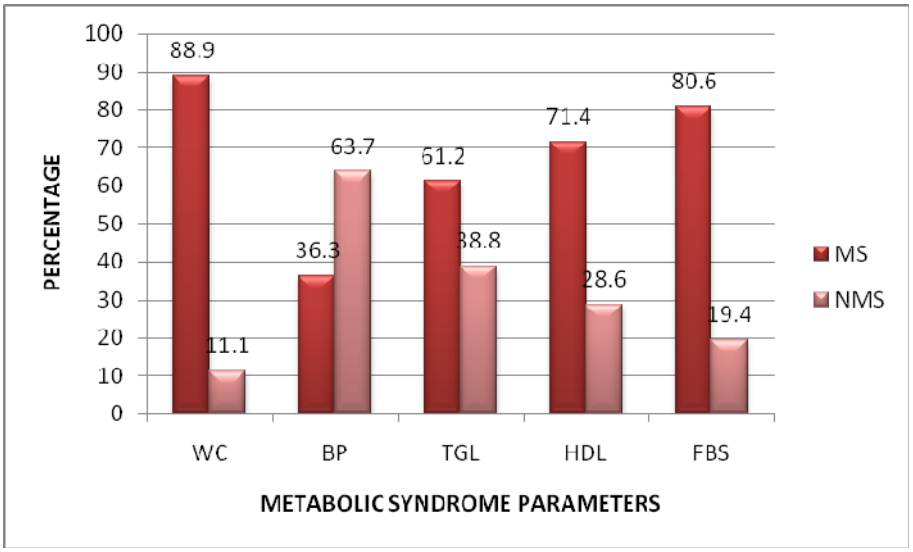


TABLE 6

COMPARATIVE EFFICACY OF THE 5 COMPONENTS.

COMPONENT OF METABOLIC SYNDROME	TRUE +VE	FALSE +VE	TRUE -VE	FALSE -VE	SENSITIVITY	SPECIFICITY	ACCURACY	PPV	NPV
BP	29	51	17	3	91	25	46	36	85
HDL	15	6	62	17	47	91	77	71	78
TGL	30	19	49	2	94	72	79	61	91
FBS	29	7	61	3	91	90	90	81	95
AC	24	3	65	8	75	96	89	89	80

Among the 5 components abdominal circumference had high specificity (96%) followed by HDL level (91%). TGL had high sensitivity (94%). Abdominal circumference also had high positive predictive value (89%).

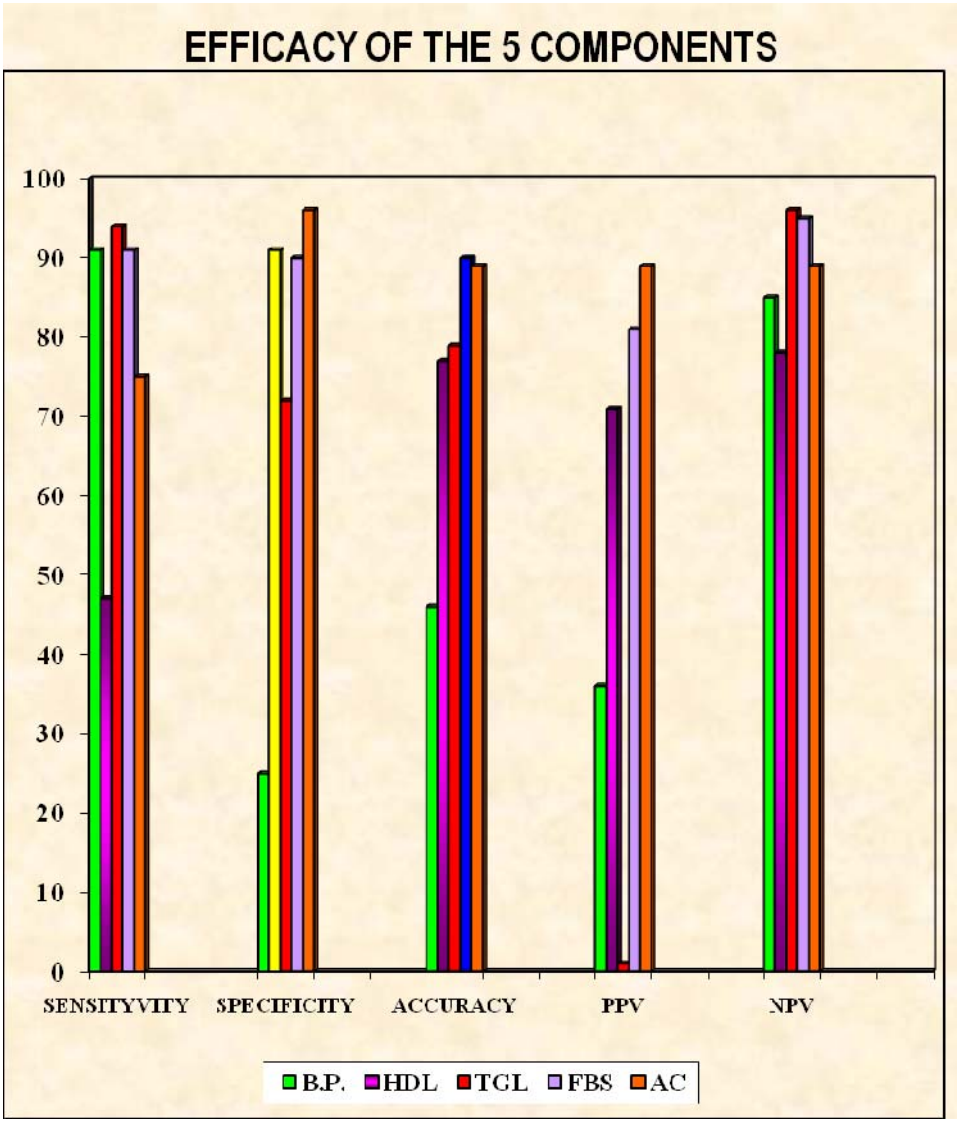


TABLE 7

DURATION OF HOSPITAL STAY

METABOLIC SYNDROME	HOSPITAL STAY		
	RANGE	MEAN	SD
POSITIVE	2 – 15	10.6	3.5
NEGATIVE	10 – 12	7.3	0.9
' p '	0.0001 (SIGNIFICANT)		

The mean duration of hospital stay was prolonged in patients with metabolic syndrome (10.6 days) compared to the control group (7.3 days). The 'p' value was 0.0001 which was statistically significant.

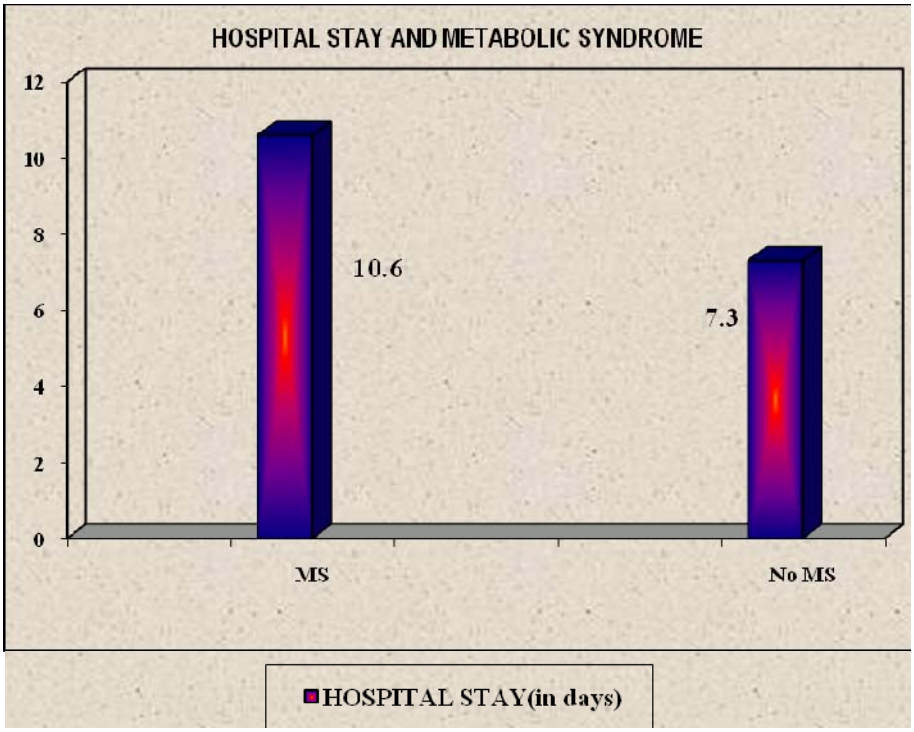


TABLE 8

EJECTION FRACTION AND METABOLIC SYNDROME.

EF	METABOLIC SYNDROME	
	POSITIVE	NEGATIVE
RANGE	30 – 42	34 – 45
MEAN	36.0	40.3
SD	3.6	2.2
' p '	0.0001 (SIGNIFICANT)	

Mean ejection fraction was low in the study group (36%) compared to the control group (40.3). The 'p' value was 0.0001 which was statistically significant.

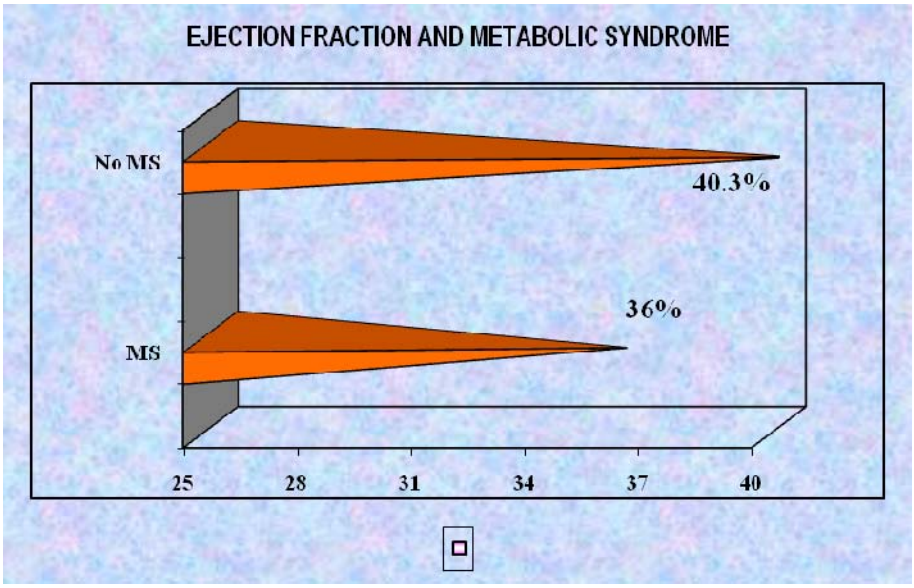


TABLE 9

KILLIP CLASS AND METABOLIC SYNDROME

KILLIP CLASS	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
1 (21)	-	-	21	100
2 (58)	20	34.5	38	65.5
3 (18)	9	50	9	50
4 (3)	3	100	-	-

Patients in the study group presented in Killip class 2 – 4. None were in killip class 1. Three patients in the study group presented in Killip class 4.

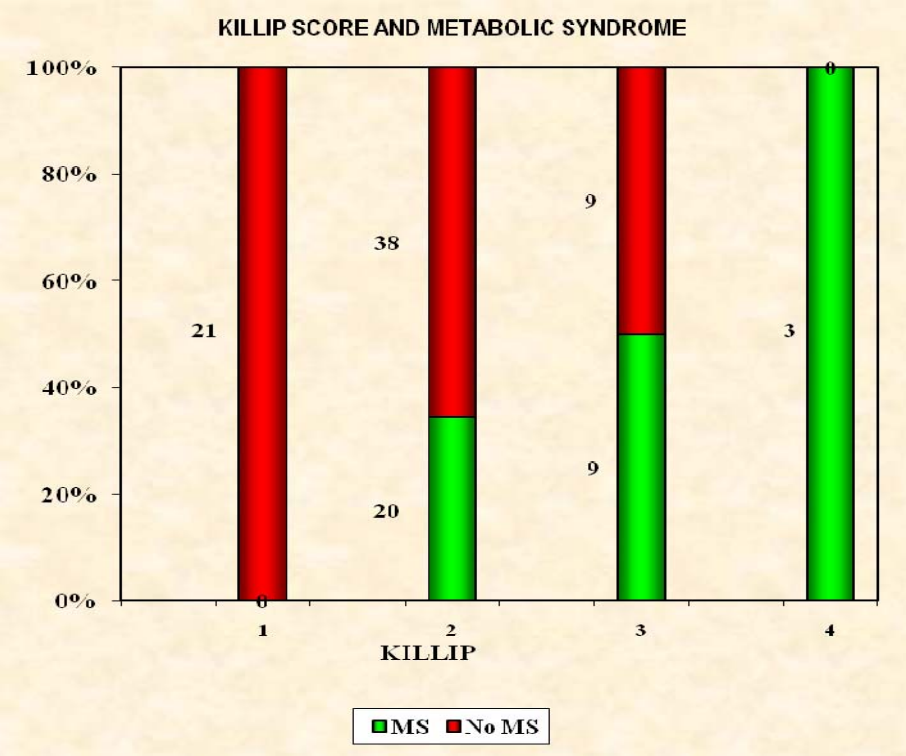


TABLE 10

STEMI / NSTEMI AND METABOLIC SYNDROME.

STEMI	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
STEMI (94)	31	33	63	67
NSTEMI (6)	1	16.7	5	83.3
' p '	0.3716 (NOT SIGNIFICANT)			

STEMI occurred in 33% of study group and 67% of control group. This was not statistically significant (p value – 0.3716).

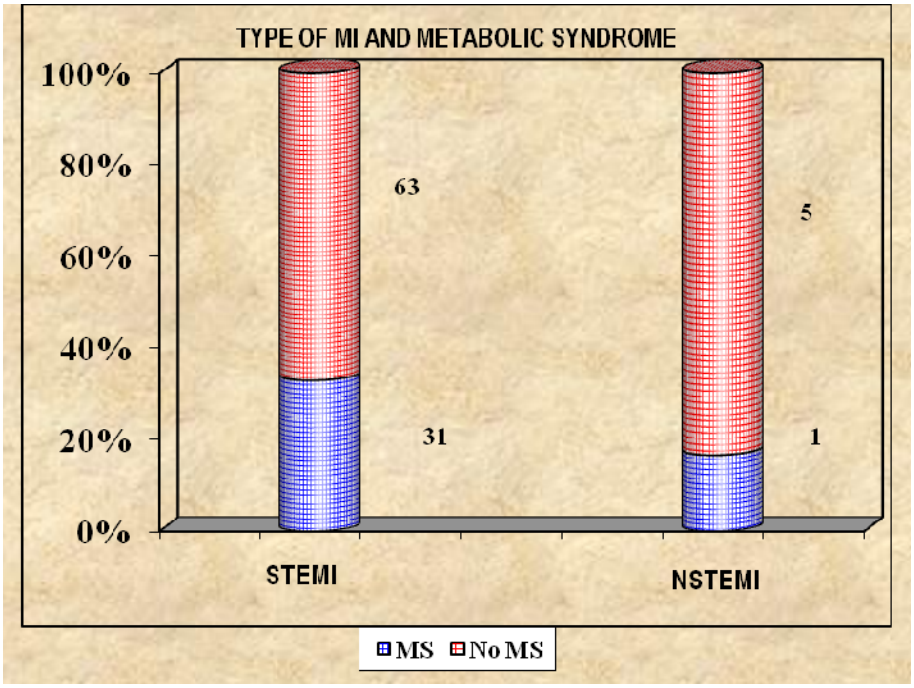


TABLE 11

THROMBOLYSIS AND METABOLIC SYNDROME.

METABOLIC SYNDROME	THROMBOLYSIS			
	YES		NO	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
POSITIVE (32)	29	90.6	3	9.4
NEGATIVE (68)	62	91.6	6	8.8
' p '	0.597 (NOT SIGNIFICANT)			

Thrombolysis was done in 90.6% of study group patients and in 91.6% of control group patients. This was not statistically significant as the 'p' value was 0.597.

TABLE 12

AGE AND METABOLIC SYNDROME.

METABOLIC SYNDROME	AGE IN YEARS		
	RANGE	MEAN	SD
POSITIVE	45 – 70	57.6	5.2
NEGATIVE	33 - 68	55.3	6.8
' p '	0.1812 (NOT SIGNIFICANT)		

It was found that metabolic syndrome patients were slightly aged (mean 57.6 years) compared to control group (mean 55.3 years), but the difference was not statistically significant.

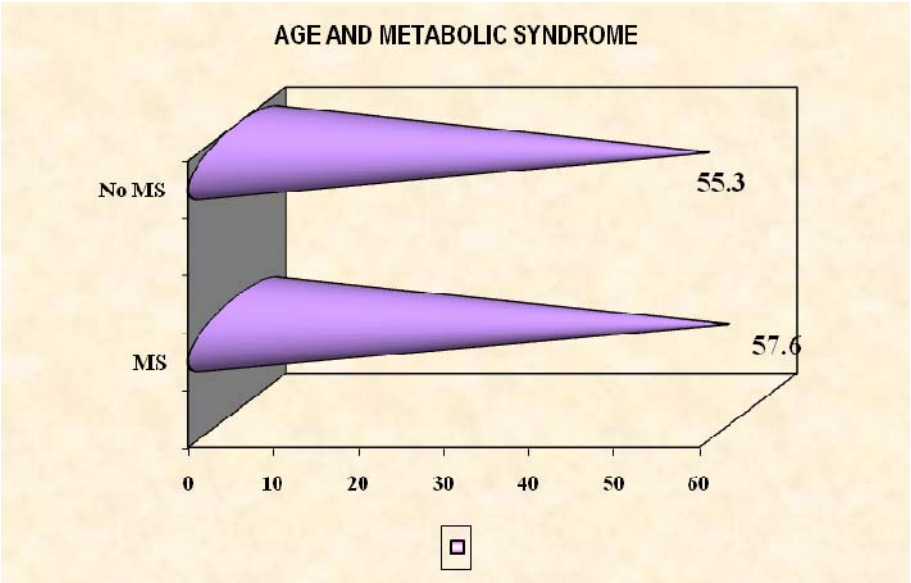


TABLE 13

GENDER AND METABOLIC SYNDROME.

SEX	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
MALES(87)	28	32.2	59	67.8
FEMALES(13)	4	30.8	9	69.2
' p '	0.5962 (NOT SIGNIFICANT)			

It was found that metabolic syndrome was more prevalent in males (32.2%) compared to females (30.8%), but the difference was not statistically significant.

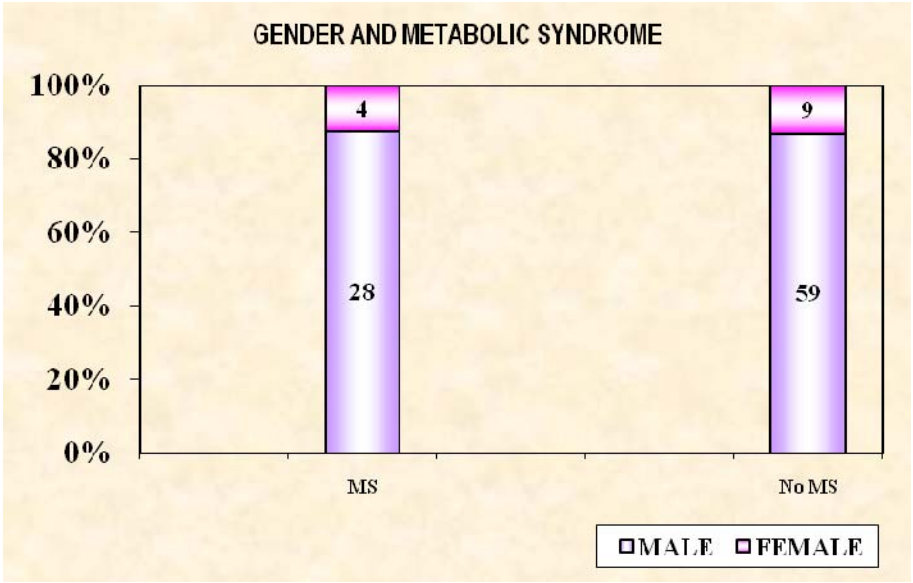


TABLE 14

AREA OF RESIDENCE AND METABOLIC SYNDROME.

RESIDENCE	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
RURAL (9)	20	29	49	71
URBAN (31)	12	38.7	19	61.3
' p '	0.464 (NOT SIGNIFICANT)			

Metabolic syndrome was slightly more prevalent in patients from urban area (38.7) compared to patients from rural area (29%), but the difference is not statistically significant (p value – 0.464).

TABLE 15

OCCUPATION AND METABOLIC SYNDROME.

OCCUPATION	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
ACTIVE(35)	5	14.3	30	85.7
SEDANTARY (65)	27	41.5	38	58.5
' p '	0.0104 SIGNIFICANT			

Among patients with metabolic syndrome 14.3% were active workers and among the control group 85.7% were active workers, the difference being statistically significant (p value – 0.0104).

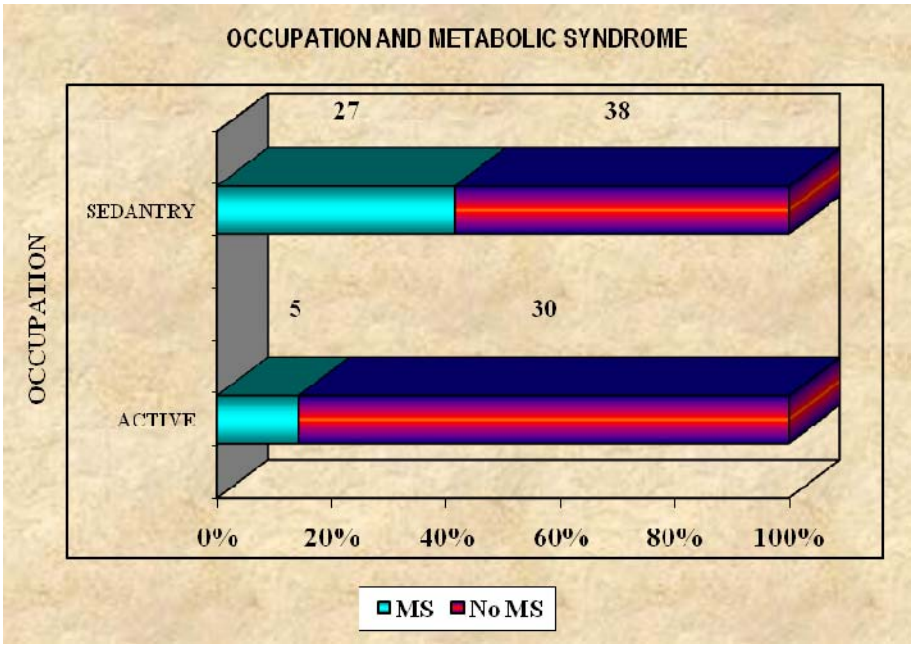


TABLE 16

EXERCISE AND METABOLIC SYNDROME.

EXERCISE	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
YES (45)	7	15.6	38	84.4
NO (55)	25	45.5	30	54.5
' p '	0.0029 (SIGNIFICANT)			

Among the study group patients, 15.6% were doing regular exercise and among the control group 84.4% were doing regular exercise. The difference is statistically significant (p value 0.0029).

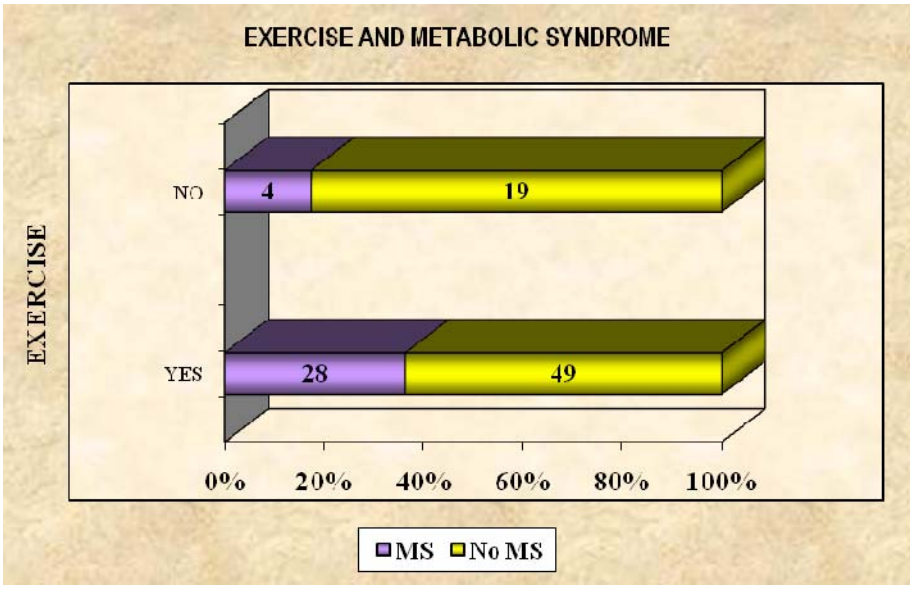


TABLE 17

FAMILY HISTORY AND METABOLIC SYNDROME.

FAMILY HISTORY	METABOLIC SYNDROME			
	POSITIVE		NEGATIVE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
YES (77)	28	36.4	49	63.6
NO (23)	4	17.4	19	82.6
' p '	0.1451 (NOT SIGNIFICANT)			

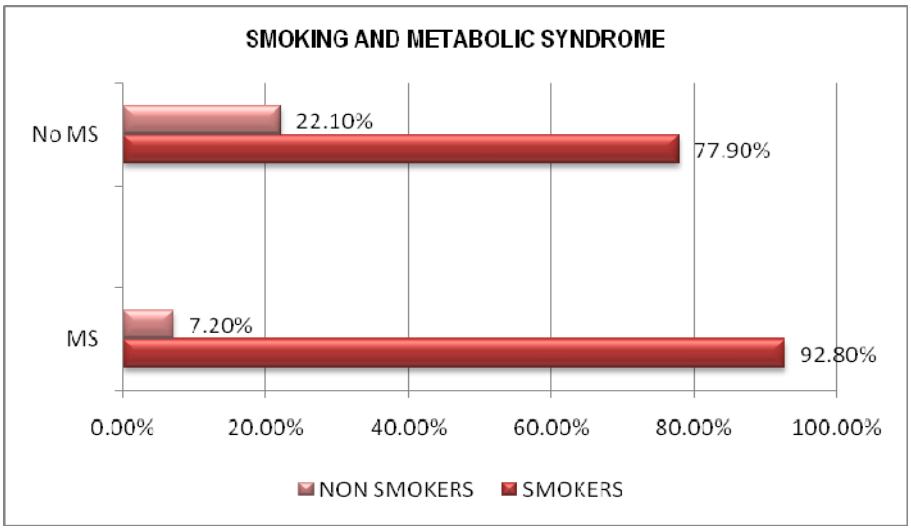
Family history was positive in 36.4% of patients in study group and 63.6% of patients in control group, the difference being not statistically significant (p value – 0.1451).

TABLE 18

SMOKING AND METABOLIC SYNDROME

SMOKING	METABOLIC SYNDROME			
	YES		NO	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
SMOKERS	26	92.8	46	77.9
NON SMOKERS	2	7.2	13	22.1
' p '	0.0738 NOT SIGNIFICANT			

Smokers were present in both groups and there was a slight increased prevalence of the syndrome in smokers, but this was not statistically significant ('p' value – 0.0738).



DISCUSSION

DISCUSSION.

100 proven cases of acute myocardial infarction who were admitted to the medical ward and cardiac intensive care unit were selected for analyzing the prevalence of metabolic syndrome. All of them satisfied the inclusion criteria.

All patients were subjected to detailed clinical history and examination. BP was recorded from all patients.12 lead ECG was done whenever indicated. ECHO was done for all patients.

Abdominal circumference of all patients were measured in standing position at a point midway between lower most point of costal margin and upper most point of iliac crest. In a few moribund patients who could not stand, measurement was taken in supine position.

Blood sample was taken for analysis of blood sugar, urea, creatinine and electrolytes on the first day and for fasting blood sugar and lipid profile on the morning of day 3.

In our study the 32 patients who satisfied the criteria for metabolic syndrome were taken as study subjects and the remaining 68 patients who had no metabolic syndrome served as the control group.

COMPARATIVE ANALYSIS

There is very limited information about the relationship of the metabolic syndrome with acute MI, particularly in South-East Asia, although western studies suggest that it is very commonly associated with coronary artery disease. A similar study was conducted by Pandey *et al*, at Nepal [2].

The prevalence of metabolic syndrome in acute MI patients in our study was 32% whereas in Pandey *et al* study it was 26.19%. but the prevalence of metabolic syndrome in the general population was 18.3% in one study conducted by Prabhdeep *et al* at Chennai (Japi June 2010). In a north Indian study the prevalence of metabolic syndrome in general population was 24.9%. the reason for a slightly higher prevalence in our study could be due to highly selected group of acutely ill patients.

The mean age of patients with metabolic syndrome is 57.6 years and about 90.6% of the study group patients were above 50 years of age. In Pandey *et al* 86% of patients were above 50 years.

COMPARISON OF THE TWO GROUPS

PARAMETERS		STUDY GROUP	CONTROL GROUP	'p' VALUE
Mean age		57.6	55.3	0.1812 (not significant)
Gender	Males	32.2%	67.8%	0.5962 (not significant)
	Females	30.8%	69.2%	
Area	Rural	29%	71%	0.464 (not significant)
	Urban	38.7%	61.3%	
Occupation	Active	14.3%	85.7%	0.0104 (significant)
	Sedentary	41.5%	58.5%	
Exercise	Yes	15.6%	84.4%	0.0029 (significant)
	No	45.5%	54.5%	
Family history	Yes	36.4%	63.6%	0.1451 (not significant)
	No	17.4%	82.6%	
Smoking	Yes	92.8%	77.9%	0.0738 (not significant)
	No	7.2%	22.1%	
MI	STEMI	33%	67%	0.3716 (not significant)
	NSTEMI	16.7%	83.3%	
Duration of stay (days)		10.6	7.3	0.0001 (significant)
EF (%)		36	40.3	0.0001 (significant)
BP	>130/85	36.3%	63.8%	0.1201 (not significant)
	<130/85	15%	85%	
HDL	Low	71.4%	28.6%	0.0001 (significant)
	High	21.5%	78.5%	
TGL	>150	61.2%	38.8%	0.0001 (significant)
	<150	3.9%	96.1%	
FBS	>110	80.6%	19.4%	0.0001 (significant)
	<110	4.7%	95.3%	
AC	High	88.9%	11.1%	0.0001 (significant)
	Low	11%	89%	

In our study, prevalence of metabolic syndrome is slightly higher in males (32.2%) compared to females (30.8%). Whereas, in Pandey *et al* study, prevalence was higher among females. A similar high prevalence among females has also been reported by Zellar *et al* [(9).]

In our study, in-hospital fatality is more among those with the metabolic syndrome (3/32) than those without the syndrome (1/68). In Pandey *et al* study similar results were obtained – in hospital fatality rate was 5/22 in patients with metabolic syndrome and 3/62 in the control group. Another study has also reported that the metabolic syndrome was associated with an increased case fatality rate. However, after adjusting for the major determinants of mortality in AMI, the metabolic syndrome was not seen to be an independent predictor of this.

Among the individual components of the metabolic syndrome, we found that increased abdominal circumference had the highest positive predictive value (89%) and this was followed by fasting blood glucose (81%); blood pressure had the least positive predictive value (36%). This indicates that obesity is associated with higher morbidity and may predispose to a higher mortality. In Pandey *et al* study, raised triglyceride levels had the highest positive predictive value (62%), followed by fasting blood sugar (55%) like our study. Blood pressure had least positive predictive value as in our study. As reported by a previous study, at long-term follow up, hypertension is only a modest predictor of death.

Duration of hospital stay is prolonged in patients in the study group (10.6 days) compared to 7.3 days in the control group. Pandey *et al* also reported a prolongation of hospital stay in their patients with metabolic syndrome. This may be due to severity of illness based on Killip class and low ejection fraction in the study group. Patients with metabolic syndrome presented with higher Killip class (> 2). None of the patients in the study group presented in class 1. Also the mean EF is reduced in the study group patients (36%) compare to control group (40.3%).

Among the study group only 15.6% were doing regular exercise and among the control group 84.4% were doing regular exercise. The difference is statistically significant. This could be the reason for the increased morbidity in the study group.

Smokers were present in both the groups. Smoking itself is an important risk factor for myocardial infarction, but there was no statistical difference between the two groups. So, smoking does not increase the morbidity associated with metabolic syndrome in myocardial infarction patients.

Among the study group only 14.3% were doing active job and among the control group 85.7% were doing active job. The difference is statistically significant.

Family history was present in 36.4% of study subjects and 63.6% of control subjects and the difference was not significant. This indicates that apart from family history, lack of exercise and sedentary job plays a role in development of metabolic syndrome.

As the prevalence of the metabolic syndrome is high worldwide and increasing day by day due to sedentary lifestyles, the findings of the present study has important implications for clinical practice. Emphasis must be placed on the intake of balanced diet and control of blood lipid levels, particularly that of triglycerides. Since elevated fasting blood glucose has such a high predictive value in acute MI, there must be a careful search for deranged carbohydrate metabolism in all cases.

SUMMARY

SUMMARY

The study “prevalence of metabolic syndrome in patients with acute myocardial infarction” was conducted in 100 acute MI patients admitted in medical wards and cardiac ICU at the Government Rajaji hospital, Madurai.

All the 100 patients satisfied the inclusion criteria and underwent various investigations – ECG, blood sugar, urea, creatinine, lipid profile and echocardiogram. Prevalence of metabolic syndrome was assessed in them by using NCEP ATP III criteria. The patients who satisfied the criteria formed the study group and those who did not formed the control group. Various parameters were compared between the two groups.

The in hospital mortality rate was high in the study group and duration of hospital stay was also high in the study group compared to the control group.

The study group had higher percentage of patients with sedentary job and physical inactivity.

The study showed increased prevalence of metabolic syndrome in acute MI patients compared to general population when compared to other studies.

CONCLUSION

CONCLUSION

The following conclusions were derived from our study,

1. The prevalence of metabolic syndrome is 32% among patients admitted with acute MI.
2. Duration of hospital stay is prolonged in patients with metabolic syndrome (10.6 days vs 7.3 days).
3. Abdominal circumference (AC) has highest positive predictive value (89%) followed by fasting blood sugar (FBS) which has (81%), followed by triglyceride (TGL) level which has (61%) positive predictive value.
4. BP has the least positive predictive value of 36%.
5. Mean EF is low in the study group (36%) compared to control group (40.3%).
6. Study group had high rate of sedentary job (41.5%) and high rate of physical inactivity (45.5%).

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BIBLIOGRAPHY

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GLOSSARY

GLOSSARY

AC	Abdominal circumference
BP	Blood pressure
CAD	Coronary artery disease
DM	Diabetes mellitus
EF	Ejection fraction
FBS	Fasting blood sugar
HTN	Hypertension
HDL	High density lipoprotein
LDL	Low density lipoprotein
MI	Myocardial infarction
MS	Metabolic syndrome
NCEP –ATPIII	National cholesterol education programme – adult treatment panel III.
NPV	Negative predictive value
NSTEMI	Non ST elevation myocardial infarction
PPV	Positive predictive value
STEMI	ST elevation myocardial infarction
TGL	Triglycerides
vWF	Von Willibrand factor

MASTER CHART

S.no	IP No	Age	Sex	Area	Occ	Exer	Smok	Alco	DM	HT	F/H	V/NV	S/N	MI	EF	KILP	Thromb	Dura	D/A	BP	HDL	TGL	FBS	AC	MS
1	254367	38	M	R	A	Y	N	N	N	N	Y	NV	S	A	40	3	Y	7	A	120/80	38	300	80	88	2
2	254376	54	M	R	S	N	Y	Y	N	N	Y	NV	S	A	38	3	Y	10	A	130/90	44	140	200	104	3
3	254373	60	M	R	A	N	Y	Y	N	N	Y	NV	S	A	36	3	Y	7	A	130/90	36	250	73	104	4
4	254372	60	M	U	S	N	Y	N	Y	Y	Y	NV	S	I	30	4	Y	2	D	130/100	40	180	130	104	4
5	254438	47	M	R	A	Y	Y	Y	N	Y	Y	NV	S	A	40	3	Y	7	A	130/90	41	240	72	83	2
6	254444	60	M	R	S	N	Y	Y	Y	Y	Y	NV	S	A	30	4	N	2	D	80/60	45	400	320	110	4
7	254448	45	M	R	S	N	Y	Y	N	N	Y	NV	S	I	36	3	Y	7	A	130/90	44	180	158	73	3
8	254451	53	M	R	A	Y	Y	Y	Y	Y	Y	NV	N	A	40	3	N	10	A	130/90	46	160	99	100	2
9	254450	52	F	R	S	N	N	N	Y	Y	Y	V	S	A	42	2	Y	7	A	140/100	48	460	336	76	4
10	254620	50	M	R	S	N	Y	Y	N	Y	Y	NV	S	A	38	3	Y	10	A	130/90	44	215	204	98	3
11	198354	70	M	U	S	N	Y	Y	Y	Y	Y	NV	S	I	38	2	Y	10	A	140/90	43	250	153	96	3
12	227233	52	M	R	A	Y	Y	Y	N	N	Y	NV	S	A	38	3	N	10	A	80/60	51	240	172	100	2
13	254736	55	F	R	S	N	N	N	Y	Y	Y	NV	S	A	40	3	Y	7	A	140/90	50	120	93	86	1
14	254740	57	M	R	A	N	Y	N	Y	Y	Y	V	S	A	42	3	Y	7	A	120/80	42	110	100	96	2
15	255732	58	M	R	A	N	N	N	Y	Y	Y	NV	S	A	42	2	Y	10	A	120/80	44	115	100	100	2
16	255781	49	F	U	S	Y	N	N	Y	N	Y	V	S	A	45	2	Y	10	A	130/90	52	115	100	80	1
17	255750	55	M	R	A	Y	Y	Y	N	Y	Y	NV	S	A	40	2	Y	7	A	140/90	45	115	102	95	2

S.no	IP No	Age	Sex	Area	Occ	Exer	Smok	Alcoh	DM	HT	F/H	V/NV	S/N	MI	EF	KILP	Thromb	Dura	D/A	BP	HDL	TGL	FBS	AC	MS
18	255741	33	M	R	S	N	Y	Y	N	N	Y	NV	N	I	38	3	N	12	A	110/70	45	100	90	80	0
19	255065	67	M	U	S	N	Y	Y	Y	Y	Y	NV	S	A	40	3	Y	10	A	140/90	40	125	124	106	3
20	255128	55	M	R	S	N	Y	Y	N	Y	Y	NV	S	A	36	3	Y	12	A	170/100	44	160	190	110	4
21	255320	52	M	R	A	Y	Y	Y	N	N	N	NV	S	I	40	3	Y	7	A	100/80	44	180	86	100	1
22	255346	62	M	R	A	N	Y	Y	Y	Y	Y	NV	S	A	38	2	Y	7	A	120/80	47	100	96	90	1
23	255434	65	M	U	A	N	Y	N	Y	Y	N	V	S	A	45	2	Y	7	D	120/80	47	110	90	90	1
24	255476	60	F	R	A	N	N	N	Y	Y	N	V	S	A	40	2	Y	7	A	110/70	51	120	100	96	2
25	255523	54	M	U	S	N	Y	N	N	Y	N	NV	S	A	42	2	Y	12	A	160/90	36	200	120	110	5
26	255579	52	M	R	A	N	Y	N	Y	N	N	V	S	A	42	1	Y	7	A	110/70	40	121	86	90	0
27	255638	52	M	R	S	N	N	N	Y	N	N	NV	S	A	38	2	Y	12	A	110/70	36	300	200	108	4
28	255674	60	M	R	A	N	Y	Y	N	Y	Y	V	S	I	38	2	Y	7	A	150/90	50	110	100	90	1
29	255689	62	F	R	A	Y	N	N	Y	Y	N	V	S	A	40	2	Y	7	A	110/70	48	110	100	86	2
30	255690	68	M	R	A	Y	Y	N	Y	Y	Y	NV	S	A	42	1	Y	7	A	110/70	40	120	100	90	1
31	255743	60	M	R	A	N	Y	N	Y	Y	N	NV	S	A	40	2	Y	12	A	150/90	40	200	160	110	4
32	255799	62	F	R	A	N	N	N	N	Y	N	V	S	A	42	1	Y	7	A	120/80	50	110	100	86	1
33	255992	57	F	R	S	Y	N	N	N	Y	Y	V	N	A	40	1	N	7	A	110/70	46	108	86	84	2
34	255999	61	M	R	A	Y	Y	Y	Y	Y	Y	V	S	I	40	2	Y	7	A	120/80	42	110	108	90	1

S.no	IP No	Age	Sex	Area	Occ	Exer	Smok	Alcoh	DM	HT	F/H	V/NV	S/N	MI	EF	KILP	Thromb	Dura	D/A	BP	HDL	TGL	FBS	AC	MS
35	256010	54	M	R	S	N	Y	N	N	N	N	V	S	A	40	2	Y	7	A	110/70	46	110	100	90	0
36	256034	60	M	R	S	N	Y	Y	Y	Y	Y	NV	S	A	32	3	Y	12	A	160/100	38	310	170	108	5
37	256067	52	M	R	A	Y	Y	N	N	Y	N	V	S	A	42	1	Y	7	A	110/70	46	110	90	86	1
38	256098	60	F	R	S	Y	N	N	Y	Y	Y	V	S	I	42	1	Y	7	A	110/70	40	110	106	85	2
39	256167	61	M	U	S	Y	Y	N	Y	Y	Y	V	S	A	40	1	Y	7	A	110/70	50	110	96	80	1
40	256198	61	M	U	S	N	Y	Y	Y	Y	Y	NV	S	I	36	2	Y	12	A	160/90	36	210	180	110	5
41	256256	40	M	R	A	N	N	N	N	N	N	NV	S	A	40	1	Y	7	A	110/70	46	120	108	110	1
42	256287	60	M	R	S	Y	Y	Y	Y	Y	Y	NV	S	A	38	2	Y	7	A	110/80	46	110	100	90	1
43	256343	58	M	R	S	N	Y	N	Y	Y	N	NV	S	A	40	2	Y	7	A	110/70	48	110	90	90	1
44	256456	62	M	R	S	N	Y	N	Y	Y	Y	V	S	A	42	2	Y	7	A	110/80	48	112	86	80	1
45	256543	60	M	U	A	N	Y	Y	Y	Y	Y	NV	N	A	36	3	N	12	A	160/100	36	310	148	108	5
46	256578	63	M	R	S	N	Y	N	Y	Y	Y	V	S	I	38	3	Y	7	A	120/80	42	111	90	86	1
47	256669	57	M	R	S	Y	Y	Y	Y	Y	Y	NV	S	A	40	2	Y	7	A	110/80	46	116	90	80	1
48	256700	57	M	R	S	N	Y	N	Y	Y	Y	NV	S	A	40	2	Y	7	A	110/74	48	108	101	81	1
49	256745	59	M	U	S	Y	Y	N	Y	Y	Y	V	S	A	42	1	Y	7	A	110/80	46	114	102	81	1
50	256750	60	M	U	A	Y	Y	Y	N	Y	Y	NV	S	A	45	1	Y	7	A	168/80	46	116	86	80	1
51	256788	54	F	R	A	Y	N	N	N	N	Y	V	S	I	45	1	Y	7	A	120/70	48	112	100	90	1

S.no	IP No	Age	Sex	Area	Occ	Exer	Smok	Alcoh	DM	HT	F/H	V/NV	S/N	MI	EF	KILP	Thromb	Dura	D/A	BP	HDL	TGL	FBS	AC	MS
52	256832	60	M	R	S	Y	N	Y	N	Y	Y	NV	S	A	40	2	Y	7	A	110/70	49	108	87	80	1
53	256846	49	M	U	S	N	Y	N	N	Y	Y	NV	S	A	40	2	Y	10	A	140/100	40	210	140	108	4
54	256877	61	M	U	S	N	Y	N	Y	Y	Y	NV	S	A	38	2	Y	12	A	120/80	36	260	108	108	3
55	256901	52	M	U	S	N	Y	N	Y	Y	Y	NV	S	A	42	1	Y	7	A	110/70	42	110	91	80	1
56	256954	61	M	R	S	Y	N	N	Y	Y	N	NV	N	A	42	2	N	7	A	110/70	48	170	101	80	2
57	256993	54	M	U	S	N	Y	N	Y	Y	Y	NV	S	A	40	2	Y	7	A	110/70	46	170	102	80	2
58	257045	61	M	R	S	N	Y	Y	Y	Y	Y	NV	S	A	30	4	N	3	D	80/60	40	300	200	108	4
59	257074	60	M	R	A	N	Y	N	N	N	Y	NV	S	A	42	2	Y	7	A	108/80	47	110	84	80	0
60	257145	49	M	U	A	Y	Y	N	N	Y	Y	V	S	A	42	2	Y	7	A	110/80	48	120	110	100	1
61	257245	48	M	U	S	N	Y	N	N	Y	Y	V	S	A	45	2	Y	7	A	108/70	46	170	108	86	2
62	257323	46	M	R	A	Y	N	N	N	Y	Y	V	S	I	40	2	Y	7	A	110/70	46	160	102	80	2
63	257368	48	M	R	A	Y	Y	N	N	N	N	NV	S	A	42	2	Y	7	A	110/80	46	170	110	100	2
64	257399	57	M	R	S	N	Y	N	Y	Y	Y	NV	S	A	42	2	Y	7	A	110/70	42	180	104	80	2
65	257412	61	M	R	A	N	Y	N	Y	Y	Y	V	S	A	42	1	Y	7	A	110/70	45	140	125	100	2
66	257450	63	M	R	A	Y	N	Y	Y	Y	Y	NV	S	A	38	2	Y	7	A	120/80	48	170	101	80	2
67	257488	53	F	U	S	Y	N	N	Y	N	Y	V	S	A	36	2	Y	7	A	110/80	41	140	90	80	0
68	257550	48	M	R	S	Y	Y	N	Y	Y	Y	V	S	A	40	2	Y	7	A	110/70	45	130	86	80	1

S.no	IP No	Age	Sex	Area	Occ	Exer	Smok	Alcoh	DM	HT	F/H	V/NV	S/N	MI	EF	KILP	Thromb	Dura	D/A	BP	HDL	TGL	FBS	AC	MS
69	257656	50	M	R	S	Y	N	Y	Y	Y	N	V	S	A	38	1	Y	7	A	110/70	42	160	96	80	2
70	257690	49	M	R	A	N	Y	N	Y	Y	Y	V	S	A	40	2	Y	7	A	120/80	44	160	101	80	2
71	257765	62	M	R	A	N	Y	N	Y	Y	Y	NV	S	A	40	1	Y	7	A	110/70	44	130	101	80	1
72	257798	57	M	R	S	N	Y	N	Y	Y	Y	NV	S	A	30	3	Y	15	A	160/90	38	300	200	96	4
73	257807	60	M	R	S	Y	Y	Y	Y	Y	Y	V	S	A	38	2	Y	12	A	120/80	42	320	180	110	4
74	257987	56	M	R	S	Y	Y	Y	Y	N	Y	V	S	A	36	2	Y	12	A	120/80	40	316	180	110	3
75	257996	67	M	R	S	Y	N	Y	Y	Y	Y	NV	S	A	40	2	Y	7	A	130/90	45	160	100	80	2
76	258110	60	F	U	S	N	N	N	Y	Y	Y	V	S	A	36	2	Y	12	A	160/100	42	300	160	88	3
77	258156	58	M	R	A	N	Y	Y	Y	Y	Y	NV	S	I	38	2	Y	7	A	110/70	46	120	100	86	1
78	258223	51	M	R	S	Y	N	N	Y	Y	Y	NV	S	A	38	2	Y	12	A	160/90	48	190	140	86	3
79	258324	48	M	U	S	Y	Y	N	Y	N	Y	NV	S	I	40	2	Y	7	A	110/70	42	160	100	89	1
80	258390	54	M	U	S	Y	N	Y	N	Y	Y	NV	S	A	36	2	Y	7	A	110/70	44	140	100	82	1
81	258426	53	M	U	S	Y	Y	N	Y	N	Y	NV	S	A	38	1	Y	7	A	110/80	44	160	102	96	1
82	258467	42	M	U	S	N	Y	N	Y	N	Y	NV	S	A	42	2	Y	7	A	120/80	46	140	180	86	1
83	258499	61	M	R	A	Y	Y	Y	Y	Y	Y	NV	S	I	30	2	Y	12	A	180/100	40	200	180	100	4
84	258523	60	M	R	S	Y	Y	N	N	Y	N	V	S	A	42	2	Y	7	A	150/80	46	140	101	87	1
85	258587	58	M	R	S	N	Y	Y	Y	Y	Y	NV	S	A	40	1	Y	7	A	140/90	44	116	140	90	1

S.no	IP No	Age	Sex	Area	Occ	Exer	Smok	Alcoh	DM	HT	F/H	V/NV	S/N	MI	EF	KILP	Thromb	Dura	D/A	BP	HDL	TGL	FBS	AC	MS
86	258623	55	M	U	S	N	N	Y	N	Y	Y	NV	S	A	36	2	Y	7	A	160/90	46	110	102	89	1
87	258650	63	M	R	S	Y	N	N	Y	N	N	NV	S	A	34	2	Y	7	A	110/70	46	160	180	90	2
88	258667	59	M	R	S	Y	Y	Y	Y	Y	Y	NV	S	I	30	2	Y	12	A	170/100	38	170	140	110	5
89	258756	60	F	U	S	Y	N	N	Y	Y	Y	NV	S	I	38	2	Y	10	A	160/90	40	160	120	90	4
90	258789	58	M	U	S	Y	Y	N	N	Y	N	NV	S	A	40	1	Y	7	A	140/100	44	110	90	86	1
91	258823	48	M	R	S	N	Y	N	Y	N	N	NV	S	A	38	1	Y	7	A	110/70	46	115	140	90	1
92	258890	56	M	U	S	Y	Y	N	N	Y	Y	V	S	A	40	1	Y	7	A	110/70	40	110	86	90	1
93	258945	60	M	R	S	N	Y	Y	N	N	N	NV	S	A	38	2	Y	7	A	110/70	38	190	90	104	3
94	259016	53	M	R	S	Y	Y	N	Y	N	Y	NV	S	I	40	2	Y	7	A	120/80	46	116	150	90	1
95	259212	52	M	U	S	N	Y	N	N	Y	Y	NV	S	A	36	2	Y	15	A	110/70	36	200	180	108	5
96	259378	60	M	U	S	N	N	N	Y	Y	N	NV	N	A	40	1	N	7	A	106/70	46	110	87	86	1
97	259456	53	M	U	S	Y	Y	N	N	Y	N	NV	S	I	38	2	Y	7	A	120/80	40	180	110	90	2
98	259498	55	M	U	A	N	Y	N	Y	Y	Y	V	S	A	36	2	Y	15	A	180/110	40	190	160	108	5
99	259557	61	F	R	S	N	N	N	Y	Y	Y	NV	S	I	34	2	Y	15	A	190/100	38	160	140	100	5
100	259592	60	M	R	S	Y	Y	Y	Y	Y	Y	NV	S	I	36	2	Y	15	A	170/100	44	186	140	110	4

AREA: R – RURAL U – URBAN

OCCUPATION (OCC): A – ACTIVE S – SEDANTARY

EXERCISE (EXER): Y – YES N – NO

SMOKING (SMOK): Y – YES N – NO

ALCOHOL (ALCO): Y – YES N – NO

DIABETES (DM): Y – YES N – NO

HYPERTENSION (HT): Y – YES N – NO

FAMILY HISTORY (F/H): Y – YES N – NO

DIET (V/NV): V –VEG NV – NON VEG

S/N: S –STEMI N – NSTEMI

MI: A – AWMI I – IWMI

EF: EJECTION FRACTION

KLIP: KILLIP CLASS

THROMBOLYSIS (THROMB): T – THROMBOLYSED N – NOT THROMBOLYSED

DURA - DURATION OF STAY

D/A – DEAD / ALIVE

MS –METABOLIC SCORE.

PROFORMA

PROFORMA

PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND ITS IMPACT ON HOSPITAL OUTCOME.

PT NO: DATE: PLACE:

PT NAME: AGE: SEX:

ADDRESS:

OCCUPATION:

PRESENT HISTORY:

PAST HISTORY: DM HTN CAD CKD CVA COPD

FAMILY HISTORY: DM HTN CAD CKD OBESITY

PERSONAL HISTORY: SMOKING: NO. OF CIGARRETE PACKS PER DAY –

NO. OF YEARS OF SMOKING –

PACK YEARS –

TOBACCO CHEWING:

ALCOHOLIC: NO.OF YEARS –

FREQUENCY -

MENSTURAL HISTORY: PRE MENOPAUSAL POST MENOPAUSAL

DIETARY HISTORY: VEG MIXED

EXAMINATION:

PR –

BP –

KILLIP CLASS –

ECG FINDINGS:

ECHO FINDIGS: EF:

TREATMENT GIVEN:

DURATION OF HOSPITAL STAY:

METABOLIC SYNDROME: (/5)

BP:	
HDL:	
TGL:	
FBS:	
ABD CIRC:	

OTHER INVESTIGATIONS:

FOLLOW – UP: